```
     FILE 'EMBASE, BIOSIS, MEDLINE, SCISEARCH, CAPLUS' ENTERED AT 10:59:56 ON
     13 JAN 2005
L1            11 S (MESSENGER RNA ANTISENSE DNA)
L2            12 S (D-RNAI) OR (DRNAI) OR (D (1W) RNAI)
L3             5 S (MRNA-CDNA INTERFER?)
L4          4990 S CHIMERIC AND OLIGONUCLEOTIDE
L5             0 S RNA/DNA AND OLIGONUCLEOTIDE
L6          4285 S RNA(1W)DNA AND OLIGONUCLEOTIDE
L7             0 S L3 AND L4
L8             0 S L3 AND L6
L9             3 S (CDNA-ARNA)
L10            1 S RNA (W) HYBRID (W) CONSTRUCT

=> s l1 and l2
L11            9 L1 AND L2

=> dup rem l11
PROCESSING COMPLETED FOR L11
L12            4 DUP REM L11 (5 DUPLICATES REMOVED)

=> d iall l12

L12  ANSWER 1 OF 4  EMBASE  COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
     on STN
ACCESSION NUMBER:    2003195139  EMBASE
TITLE:               Erratum: D-RNAi (messenger
                     RNA-antisense DNA interference)
                     as a novel defense system against cancer and viral
                     infections (Current Cancer Drug Targets (2001) 1
                     (241-247)).
AUTHOR:              Lin S.-L.; Ying S.-Y.
SOURCE:              Current Cancer Drug Targets, (2003) 3/3 (237).
                     ISSN: 1568-0096  CODEN: CCDTB
COUNTRY:             Netherlands
DOCUMENT TYPE:       Journal; Errata
FILE SEGMENT:        016     Cancer
LANGUAGE:            English

CONTROLLED TERM:     Medical Descriptors:
                     *error
                     erratum


=> d iall l12 2-4

L12  ANSWER 2 OF 4  CAPLUS  COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER:         2003:383764  CAPLUS
DOCUMENT NUMBER:          140:156350
ENTRY DATE:               Entered STN:  20 May 2003
TITLE:                    D-RNAi (messenger
                          RNA-antisense DNA
                          interference) as a novel defense system against cancer
                          and viral infections. [Erratum to document cited in
                          CA136:128434]
AUTHOR(S):                Lin, Shi-Lung; Suksaweang, Sanong; Chuong, Cheng-Ming;
                          Rasheed Suraiya; Ying, Shao-Yao
CORPORATE SOURCE:         Epiclone, Inc., Alhambra, CA, 91801, USA
SOURCE:                   Current Cancer Drug Targets (2003), 3(3), 237
                          CODEN: CCDTB9; ISSN: 1568-0096
PUBLISHER:                Bentham Science Publishers Ltd.
DOCUMENT TYPE:            Journal; General Review
LANGUAGE:                 English
```

CLASSIFICATION: 1-0 (Pharmacology)
Section cross-reference(s): 13

ABSTRACT:
A review. On page 241, line 3, the names of Sanong Suksaweang, Cheng-Ming Chuong, and Suraiya Rasheed are added as the second, third, and fourth authors and the corrected affiliations (page 241, lines 4-6) are given. Shi-Lung Lin is the only author affiliated with Epiclone Inc., San Diego, CA, USA 92130. Shi-Lung Lin, Sanong Suksaweang, and Cheng-Ming Chuong are affiliated with the Department of Pathol., Keck School of Medicine, University of Southern California, HMR-209, 2011 Zonal Avenue, Los Angeles, CA USA 90033. Suraiya Rasheed is affiliated with the Laboratory of Viral Oncol. and AIDS Research, Department of Pathol., Keck School of Medicine, University of Southern California, Los Angeles CA 90032-3626. Shao-Yao Ying is affiliated with the Department of Cell and Neurobiol., Keck School of Medicine, BMT-401, University of Southern California, 1333 San Pablo Street, Los Angeles, CA 90033. In Figure 3A on page 244, the albumin should be GAPDH.

SUPPL. TERM: erratum review RNA antisense DNA hybrid gene knockout; cancer treatment mRNA antisense DNA hybrid review erratum; viral infection mRNA antisense DNA hybrid review erratum

INDEX TERM: Antitumor agents
Antiviral agents
(**D-RNAi** (mRNA-antisense DNA interference) for posttranscriptional gene knockout as novel defense system against cancer and viral infections (Erratum))

INDEX TERM: Gene
ROLE: BSU (Biological study, unclassified); BIOL (Biological study)
(expression; **D-RNAi** (mRNA-antisense DNA interference) for posttranscriptional gene knockout as novel defense system against cancer and viral infections (Erratum))

INDEX TERM: Gene targeting
(gene knock-out; **D-RNAi** (mRNA-antisense DNA interference) for posttranscriptional gene knockout as novel defense system against cancer and viral infections (Erratum))

INDEX TERM: mRNA
ROLE: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hybrids with antisense DNA; **D-RNAi** (mRNA-antisense DNA interference) for posttranscriptional gene knockout as novel defense system against cancer and viral infections (Erratum))

INDEX TERM: Antisense DNA
ROLE: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hybrids with mRNA; **D-RNAi** (mRNA-antisense DNA interference) for posttranscriptional gene knockout as novel defense system against cancer and viral infections (Erratum))

L12 ANSWER 3 OF 4 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 1

ACCESSION NUMBER: 2002338876 EMBASE
TITLE: Regulation of cell proliferation, apoptosis, and carcinogenesis by activin.
AUTHOR: Chen Y.-G.; Lui H.M.; Lin S.-L.; Lee J.M.; Ying S.-Y.
CORPORATE SOURCE: S.-Y. Ying, Department of Neurobiology, Keck School of

Medicine, University of Southern California, 1333 San Pablo Street (BMT-401), Los Angeles, CA 90089-9112, United States. sying@hsc.use.edu
SOURCE: Experimental Biology and Medicine, (2002) 227/2 (75-87).
Refs: 180
ISSN: 0037-9727 CODEN: EBMMBE
COUNTRY: United States
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 016 Cancer
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT:
The aim of this review is to provide insight into the molecular mechanisms by which activin A modulates cell proliferation, apoptosis, and carcinogenesis in vitro and in vivo. Activin A, a member of the TGFβ superfamily, has various effects on diverse biological systems, including cell growth inhibition in many cell types. However, the mechanism(s) by which activin exerts its inhibitory effects are not yet understood. This review highlights activin's effects on activin receptors and signaling pathway, modulation of activin signaling, and regulation of cell proliferation and apoptosis by activin. Based on the experiences of all the authors, we emphasized cell cycle inhibitors such as p16 and p21 and regulators of apoptosis such as p53 and members of the bcl-2 family. Aside from activin's inhibition of cell proliferation and enhancement of apoptosis, other newly developed methods for molecular studies of apoptosis by activin were briefly presented that support the role of activin as an inhibitor of carcinogenesis and cancer progression. These methods include subtractive hybridization based on covalent bonding, a simple and accurate means to determine molecular profile of as few as 20 cells based on an RNA-PCR approach, and a **messenger RNA-antisense** ***DNA*** interference phenomenon (**D-RNAi**), resulting in a long-term gene knockout effects.

CONTROLLED TERM: Medical Descriptors:
*cell proliferation
*apoptosis
*carcinogenesis
cell growth
cancer growth
covalent bond
polymerase chain reaction
protein function
signal transduction
knockout gene
human
nonhuman
short survey
Drug Descriptors:
*activin A: EC, endogenous compound
*transforming growth factor beta
*activin receptor: EC, endogenous compound
protein p16
protein p21
protein p53
protein bcl 2
CAS REGISTRY NO.: (activin A) 104625-48-1; (protein p21) 85306-28-1; (protein bcl 2) 219306-68-0

ACCESSION NUMBER: 2003:44652 BIOSIS
DOCUMENT NUMBER: PREV200300044652

TITLE: **D-RNAi (Messenger RNA -antisense DNA** interference) as a novel defense system against cancer and viral infections.

AUTHOR(S): Lin, Shi-Lung; Ying, Shao-Yao [Reprint Author]
CORPORATE SOURCE: Department of Cell and Neurobiology, Keck School of Medicine, University of Southern California, 1333 San Pablo Street, BMT-401, Los Angeles, CA, 90033, USA
sying@hsc.usc.edu

SOURCE: Current Cancer Drug Targets, (November 2001) Vol. 1, No. 3, pp. 241-247. print.
ISSN: 1568-0096 (ISSN print).
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Jan 2003
Last Updated on STN: 15 Jan 2003

CONCEPT CODE:
Genetics - General 03502
Genetics - Animal 03506
Genetics - Human 03508
Biochemistry studies - General 10060
Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
Biochemistry studies - Proteins, peptides and amino acids 10064
Neoplasms - Pathology, clinical aspects and systemic effects 24004
Development and Embryology - General and descriptive 25502
Genetics of bacteria and viruses 31500
Virology - General and methods 33502
Medical and clinical microbiology - Virology 36006

INDEX TERMS:
Major Concepts
Infection; Molecular Genetics (Biochemistry and Molecular Biophysics); Tumor Biology

INDEX TERMS:
Diseases
cancer: neoplastic disease
Neoplasms (MeSH)

INDEX TERMS:
Diseases
viral infections: viral disease
Virus Diseases (MeSH)

INDEX TERMS:
Chemicals & Biochemicals
antisense DNA; bcl-2; messenger RNA; phorbol ester

INDEX TERMS:
Miscellaneous Descriptors
apoptosis; gene silencing; **messenger RNA-antisense DNA** interference

ORGANISM:
Classifier
Galliformes 85536
Super Taxa
Aves; Vertebrata; Chordata; Animalia
Organism Name
chicken (common): embryo, animal model
Taxa Notes
Animals, Birds, Chordates, Nonhuman Vertebrates, Vertebrates

ORGANISM:
Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
H9 cell line (cell line): human CD4-positive T cell
LNCaP cell line (cell line): human prostate cancer cell

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                    Taxa Notes
                       Animals, Chordates, Humans, Mammals, Primates,
                       Vertebrates
ORGANISM:           Classifier
                       Retroviridae   03305
                    Super Taxa
                       DNA and RNA Reverse Transcribing Viruses; Viruses;
                       Microorganisms
                    Organism Name
                       HIV-1 (miscellaneous) [Human immunodeficiency virus 1
                       (species)]: pathogen
                    Taxa Notes
                       DNA and RNA Reverse Transcribing Viruses,
                       Microorganisms, Viruses
GENE NAME:          beta-catenin gene


=> FIL STNGUIDE
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                     76.44      77.70

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LAST RELOADED: Jan  7, 2005 (20050107/UP).

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COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                      0.12      77.82

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                      0.00      -0.73

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PROCESSING COMPLETED FOR L2
L13             5 DUP REM L2 (7 DUPLICATES REMOVED)

=> d iall l13 1-5
```

L13 ANSWER 1 OF 5 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 2003195139 EMBASE
TITLE: Erratum: **D-RNAi** (messenger RNA-antisense DNA interference) as a novel defense system against cancer and viral infections (Current Cancer Drug Targets (2001) 1 (241-247)).
AUTHOR: Lin S.-L.; Ying S.-Y.
SOURCE: Current Cancer Drug Targets, (2003) 3/3 (237).
ISSN: 1568-0096 CODEN: CCDTB
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Errata
FILE SEGMENT: 016 Cancer
LANGUAGE: English

CONTROLLED TERM: Medical Descriptors:
*error
erratum

L13 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:383764 CAPLUS
DOCUMENT NUMBER: 140:156350
ENTRY DATE: Entered STN: 20 May 2003
TITLE: **D-RNAi** (messenger RNA-antisense DNA interference) as a novel defense system against cancer and viral infections. [Erratum to document cited in CA136:128434]
AUTHOR(S): Lin, Shi-Lung; Suksaweang, Sanong; Chuong, Cheng-Ming; Rasheed Suraiya; Ying, Shao-Yao
CORPORATE SOURCE: Epiclone, Inc., Alhambra, CA, 91801, USA
SOURCE: Current Cancer Drug Targets (2003), 3(3), 237
CODEN: CCDTB9; ISSN: 1568-0096
PUBLISHER: Bentham Science Publishers Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
CLASSIFICATION: 1-0 (Pharmacology)
Section cross-reference(s): 13

ABSTRACT:
A review. On page 241, line 3, the names of Sanong Suksaweang, Cheng-Ming Chuong, and Suraiya Rasheed are added as the second, third, and fourth authors and the corrected affiliations (page 241, lines 4-6) are given. Shi-Lung Lin is the only author affiliated with Epiclone Inc., San Diego, CA, USA 92130. Shi-Lung Lin, Sanong Suksaweang, and Cheng-Ming Chuong are affiliated with the Department of Pathol., Keck School of Medicine, University of Southern California, HMR-209, 2011 Zonal Avenue, Los Angeles, CA USA 90033. Suraiya Rasheed is affiliated with the Laboratory of Viral Oncol. and AIDS Research, Department of Pathol., Keck School of Medicine, University of Southern California, Los Angeles CA 90032-3626. Shao-Yao Ying is affiliated with the Department of Cell and Neurobiol., Keck School of Medicine, BMT-401, University of Southern California, 1333 San Pablo Street, Los Angeles, CA 90033. In Figure 3A on page 244, the albumin should be GAPDH.

SUPPL. TERM: erratum review RNA antisense DNA hybrid gene knockout; cancer treatment mRNA antisense DNA hybrid review erratum; viral infection mRNA antisense DNA hybrid review erratum
INDEX TERM: Antitumor agents
Antiviral agents
(**D-RNAi** (mRNA-antisense DNA interference) for posttranscriptional gene knockout as novel defense system against cancer and viral infections (Erratum))

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ROLE: BSU (Biological study, unclassified); BIOL (Biological study)
(expression; **D-RNAi** (mRNA-antisense DNA interference) for posttranscriptional gene knockout as novel defense system against cancer and viral infections (Erratum))

INDEX TERM: Gene targeting
(gene knock-out; **D-RNAi** (mRNA-antisense DNA interference) for posttranscriptional gene knockout as novel defense system against cancer and viral infections (Erratum))

INDEX TERM: mRNA
ROLE: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hybrids with antisense DNA; **D-RNAi** (mRNA-antisense DNA interference) for posttranscriptional gene knockout as novel defense system against cancer and viral infections (Erratum))

INDEX TERM: Antisense DNA
ROLE: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hybrids with mRNA; **D-RNAi** (mRNA-antisense DNA interference) for posttranscriptional gene knockout as novel defense system against cancer and viral infections (Erratum))

L13 ANSWER 3 OF 5 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 1

ACCESSION NUMBER: 2002338876 EMBASE
TITLE: Regulation of cell proliferation, apoptosis, and carcinogenesis by activin.
AUTHOR: Chen Y.-G.; Lui H.M.; Lin S.-L.; Lee J.M.; Ying S.-Y.
CORPORATE SOURCE: S.-Y. Ying, Department of Neurobiology, Keck School of Medicine, University of Southern California, 1333 San Pablo Street (BMT-401), Los Angeles, CA 90089-9112, United States. sying@hsc.use.edu
SOURCE: Experimental Biology and Medicine, (2002) 227/2 (75-87). Refs: 180
ISSN: 0037-9727 CODEN: EBMMBE
COUNTRY: United States
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 016 Cancer
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT:
The aim of this review is to provide insight into the molecular mechanisms by which activin A modulates cell proliferation, apoptosis, and carcinogenesis in vitro and in vivo. Activin A, a member of the TGFβ superfamily, has various effects on diverse biological systems, including cell growth inhibition in many cell types. However, the mechanism(s) by which activin exerts its inhibitory effects are not yet understood. This review highlights activin's effects on activin receptors and signaling pathway, modulation of activin signaling, and regulation of cell proliferation and apoptosis by activin. Based on the experiences of all the authors, we emphasized cell cycle inhibitors such as p16 and p21 and regulators of apoptosis such as p53 and members of the bcl-2 family. Aside from activin's inhibition of cell proliferation and enhancement of apoptosis, other newly developed methods for molecular studies of apoptosis by activin were briefly presented that support the role of activin as an

inhibitor of carcinogenesis and cancer progression. These methods include subtractive hybridization based on covalent bonding, a simple and accurate means to determine molecular profile of as few as 20 cells based on an RNA-PCR approach, and a messenger RNA-antisense DNA interference phenomenon (**D -RNAi**), resulting in a long-term gene knockout effects.

CONTROLLED TERM: Medical Descriptors:
*cell proliferation
*apoptosis
*carcinogenesis
cell growth
cancer growth
covalent bond
polymerase chain reaction
protein function
signal transduction
knockout gene
human
nonhuman
short survey
Drug Descriptors:
*activin A: EC, endogenous compound
*transforming growth factor beta
*activin receptor: EC, endogenous compound
protein p16
protein p21
protein p53
protein bcl 2

CAS REGISTRY NO.: (activin A) 104625-48-1; (protein p21) 85306-28-1; (protein bcl 2) 219306-68-0

L13 ANSWER 4 OF 5 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN DUPLICATE 2

ACCESSION NUMBER: 2001:234339 SCISEARCH
THE GENUINE ARTICLE: 409XJ
TITLE: A novel mRNA-cDNA interference phenomenon for silencing bcl-2 expression in human LNCaP cells
AUTHOR: Lin S L; Chuong C M (Reprint); Ying S Y
CORPORATE SOURCE: Univ So Calif, Keck Sch Med, Dept Pathol, HMR 209, 2011 Zonal Ave, Los Angeles, CA 90033 USA (Reprint); Univ So Calif, Keck Sch Med, Dept Pathol, Los Angeles, CA 90033 USA; Univ So Calif, Keck Sch Med, Dept Cell & Neurobiol, Los Angeles, CA 90033 USA; Epiclone Inc, Alhambra, CA 91801 USA
COUNTRY OF AUTHOR: USA
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (2 MAR 2001) Vol. 281, No. 3, pp. 639-644.
Publisher: ACADEMIC PRESS INC, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495 USA.
ISSN: 0006-291X.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 26
ABSTRACT:

The templates required for inducing posttranscriptional gene silencing (PTGS) effects have been investigated in human prostate cancer LNCaP cells. Transfection of a mRNA-cDNA hybrid construct was found to result in a relatively long-term interference of specific gene expression. Androgen-stimulated expression of bcl-2 has been reported to increase the tumorigenic and metastatic potentials of human prostate cancer LNCaP cells, as well as their resistance to many apoptotic stimuli. The addition of bcl-2 antisense oligonucleotides, however, restored apoptosis. Our studies

demonstrate gene silencing effects of the mRNA-cDNA transfection that is similar to those of PTGS/RNAi in this in vitro prostate cancer cell model. A potential RNA-directed RNA polymerase activity was also detected which is alpha -amanitin-sensitive. These findings indicate that a novel gene silencing system may existinmammaliancells. (C) 2001 Academic Press.

CATEGORY: BIOCHEMISTRY & MOLECULAR BIOLOGY; BIOPHYSICS
SUPPLEMENTARY TERM: mRNA-cDNA interference phenomenon; (**D-RNAi**); posttranscriptional gene silencing (PTGS); RNA-directed RNA polymerase (RaRp); prostate cancer cells; bcl-2
SUPPL. TERM PLUS: DOUBLE-STRANDED-RNA; PROSTATE-CANCER CELLS; C-ELEGANS; IN-VIVO; MESSENGER-RNA; GENE-FUNCTION; APOPTOSIS; RESISTANCE; POLYMERASE; DROSOPHILA

REFERENCE(S):

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	ARN PG (RPG)	Referenced Work (RWK)
BAULCOMBE D C	2000	290	1108	SCIENCE
BERCHEM G J	1995	55	735	CANCER RES
BOSHER J M	2000	2	31	NAT CELL BIOL
COGONI C	1999	399	166	NATURE
COLOMBEL M	1993	143	390	AM J PATHOL
FILIPOVSKA J	2000	6	41	RNA
FIRE A	1998	391	806	NATURE
GRANT S R	1999	96	303	CELL
GRISHOK A	2000	287	2494	SCIENCE
HSIAO M	1997	233	359	BIOCHEM BIOPH RES CO
KETTING R F	1999	99	133	CELL
LIN S L	1999	27	4585	NUCLEIC ACIDS RES
LIN S L	1999	257	187	BIOCHEM BIOPH RES CO
MCCONKEY D J	1996	56	5594	CANCER RES
MISQUITTA L	1999	96	1451	P NATL ACAD SCI USA
MODAHL L E	2000	20	6030	MOL CELL BIOL
PALBHADRA M	1999	99	35	CELL
RAFFO A J	1995	55	4438	CANCER RES
REED J C	1990	50	6565	CANCER RES
SAMBROOK J	1989			MOL CLONING LAB MANU
SMARDON A	2000	10	169	CURR BIOL
TABARA H	1999	99	123	CELL
WARGELIUS A	1999	263	156	BIOCHEM BIOPH RES CO
WIANNY F	2000	2	70	NAT CELL BIOL
YANG D	2000	10	1191	CURR BIOL
ZAMORE P D	2000	101	25	CELL

L13 ANSWER 5 OF 5 DUPLICATE 3 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2003:44652 BIOSIS
DOCUMENT NUMBER: PREV200300044652
TITLE: **D-RNAi** (Messenger RNA-antisense DNA interference) as a novel defense system against cancer and viral infections.
AUTHOR(S): Lin, Shi-Lung; Ying, Shao-Yao [Reprint Author]
CORPORATE SOURCE: Department of Cell and Neurobiology, Keck School of Medicine, University of Southern California, 1333 San Pablo Street, BMT-401, Los Angeles, CA, 90033, USA
sying@hsc.usc.edu
SOURCE: Current Cancer Drug Targets, (November 2001) Vol. 1, No. 3, pp. 241-247. print.
ISSN: 1568-0096 (ISSN print).
DOCUMENT TYPE: Article
General Review; (Literature Review)

LANGUAGE: English
ENTRY DATE: Entered STN: 15 Jan 2003
Last Updated on STN: 15 Jan 2003
CONCEPT CODE: Genetics - General 03502
Genetics - Animal 03506
Genetics - Human 03508
Biochemistry studies - General 10060
Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
Biochemistry studies - Proteins, peptides and amino acids 10064
Neoplasms - Pathology, clinical aspects and systemic effects 24004
Development and Embryology - General and descriptive 25502
Genetics of bacteria and viruses 31500
Virology - General and methods 33502
Medical and clinical microbiology - Virology 36006
INDEX TERMS: Major Concepts
Infection; Molecular Genetics (Biochemistry and Molecular Biophysics); Tumor Biology
INDEX TERMS: Diseases
cancer: neoplastic disease
Neoplasms (MeSH)
INDEX TERMS: Diseases
viral infections: viral disease
Virus Diseases (MeSH)
INDEX TERMS: Chemicals & Biochemicals
antisense DNA; bcl-2; messenger RNA; phorbol ester
INDEX TERMS: Miscellaneous Descriptors
apoptosis; gene silencing; messenger RNA-antisense DNA interference
ORGANISM: Classifier
Galliformes 85536
Super Taxa
Aves; Vertebrata; Chordata; Animalia
Organism Name
chicken (common): embryo, animal model
Taxa Notes
Animals, Birds, Chordates, Nonhuman Vertebrates, Vertebrates
ORGANISM: Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
H9 cell line (cell line): human CD4-positive T cell
LNCaP cell line (cell line): human prostate cancer cell
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
ORGANISM: Classifier
Retroviridae 03305
Super Taxa
DNA and RNA Reverse Transcribing Viruses; Viruses; Microorganisms
Organism Name
HIV-1 (miscellaneous) [Human immunodeficiency virus 1 (species)]: pathogen
Taxa Notes
DNA and RNA Reverse Transcribing Viruses, Microorganisms, Viruses

GENE NAME: beta-catenin gene

=> d iall l3 1-5

L3 ANSWER 1 OF 5 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
ACCESSION NUMBER: 2001347960 EMBASE
TITLE: A novel **mRNA-cDNA interference** phenomenon for silencing bcl-2 expression in human LNCaP cells.
AUTHOR: Lin S.L.; Chuong C.M.; Ying S.Y.
CORPORATE SOURCE: C.M. Chuong, Department of Pathology, Keck School of Medicine, University of Southern California, 2011 Zonal Avenue, Los Angeles, CA 90033, United States. chuong@pathfinder.hsc.usc.edu
SOURCE: Biochemical and Biophysical Research Communications, (2001) 281/3 (639-644).
Refs: 27
ISSN: 0006-291X CODEN: BBRCA
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT:
The templates required for inducing posttranscriptional gene silencing (PTGS) effects have been investigated in human prostate cancer LNCaP cells. Transfection of a mRNA-cDNA hybrid construct was found to result in a relatively long-term interference of specific gene expression. Androgen-stimulated expression of bcl-2 has been reported to increase the tumorigenic and metastatic potentials of human prostate cancer LNCaP cells, as well as their resistance to many apoptotic stimuli. The addition of bcl-2 antisense oligonucleotides, however, restored apoptosis. Our studies demonstrate gene silencing effects of the mRNA-cDNA transfection that is similar to those of PTGS/RNAi in this in vitro prostate cancer cell model. A potential RNA-directed RNA polymerase activity was also detected which is α-amanitin-sensitive. These findings indicate that a novel gene silencing system may exist in mammalian cells. .COPYRGT. 2001 Academic Press.

CONTROLLED TERM: Medical Descriptors:
*gene silencing
DNA template
prostate cancer
cancer cell culture
genetic transfection
gene expression
carcinogenesis
mutagenesis
apoptosis
enzyme activity
human
controlled study
human cell
article
priority journal
Drug Descriptors:
*messenger RNA
*complementary DNA
*protein bcl 2: EC, endogenous compound
antisense oligonucleotide

RNA directed RNA polymerase
amanitin

CAS REGISTRY NO.: (protein bcl 2) 219306-68-0; (RNA directed RNA polymerase) 9026-28-2; (amanitin) 11030-71-0

L3 ANSWER 2 OF 5 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2001:207415 BIOSIS
DOCUMENT NUMBER: PREV200100207415
TITLE: A novel **mRNA-cDNA interference** phenomenon for silencing bcl-2 expression in human LNCaP cells.
AUTHOR(S): Lin, Shi-Lung; Chuong, Cheng-Ming [Reprint author]; Ying, Shao-Yao
CORPORATE SOURCE: Department of Pathology, Keck School of Medicine, University of Southern California, 2011 Zonal Avenue, HMR-209, Los Angeles, CA, 90033, USA
chuong@pathfinder.hsc.usc.edu; sying@usc.edu
SOURCE: Biochemical and Biophysical Research Communications, (March 2, 2001) Vol. 281, No. 3, pp. 639-644. print.
CODEN: BBRCA9. ISSN: 0006-291X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Apr 2001
Last Updated on STN: 18 Feb 2002

ABSTRACT:The templates required for inducing posttranscriptional gene silencing (PTGS) effects have been investigated in human prostate cancer LNCaP cells. Transfection of a mRNA-cDNA hybrid construct was found to result in a relatively long-term interference of specific gene expression. Androgen-stimulated expression of bcl-2 has been reported to increase the tumorigenic and metastatic potentials of human prostate cancer LNCaP cells, as well as their resistance to many apoptotic stimuli. The addition of bcl-2 antisense oligonucleotides, however, restored apoptosis. Our studies demonstrate gene silencing effects of the mRNA-cDNA transfection that is similar to those of PTGS/RNAi in this in vitro prostate cancer cell model. A potential RNA-directed RNA polymerase activity was also detected which is alpha-amanitin-sensitive. These findings indicate that a novel gene silencing system may exist in mammalian cells.

CONCEPT CODE: Biochemistry studies - Proteins, peptides and amino acids 10064
Cytology - General 02502
Cytology - Animal 02506
Cytology - Human 02508
Genetics - General 03502
Genetics - Animal 03506
Genetics - Human 03508
Biochemistry studies - General 10060
Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
Enzymes - General and comparative studies: coenzymes 10802
Neoplasms - Pathology, clinical aspects and systemic effects 24004

INDEX TERMS: Major Concepts
Biochemistry and Molecular Biophysics; Molecular Genetics (Biochemistry and Molecular Biophysics); Cell Biology; Tumor Biology

INDEX TERMS: Chemicals & Biochemicals
RNA polymerase: RNA directed; bcl-2: androgen-stimulated expression, expression; cDNA [complementary DNA]; mRNA [messenger RNA]

INDEX TERMS: Miscellaneous Descriptors
posttranscriptional gene silencing: effect

ORGANISM: Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
LNCaP cell line: human prostate cancer cells, model
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
ORGANISM: Classifier
Mammalia 85700
Super Taxa
Vertebrata; Chordata; Animalia
Organism Name
mammal
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
REGISTRY NUMBER: 9014-24-8 (RNA polymerase)

L3 ANSWER 3 OF 5 MEDLINE on STN
ACCESSION NUMBER: 2001216119 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11237705
TITLE: A Novel **mRNA-cDNA interference** phenomenon for silencing bcl-2 expression in human LNCaP cells.
AUTHOR: Lin S L; Chuong C M; Ying S Y
CORPORATE SOURCE: Department of Pathology, Keck School of Medicine, University of Southern California, HMR-209, 2011 Zonal Avenue, Los Angeles, California, 90033, USA.
SOURCE: Biochemical and biophysical research communications, (2001 Mar 2) 281 (3) 639-44.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 20010425
Last Updated on STN: 20010425
Entered Medline: 20010419

ABSTRACT:
The templates required for inducing posttranscriptional gene silencing (PTGS) effects have been investigated in human prostate cancer LNCaP cells. Transfection of a mRNA-cDNA hybrid construct was found to result in a relatively long-term interference of specific gene expression. Androgen-stimulated expression of bcl-2 has been reported to increase the tumorigenic and metastatic potentials of human prostate cancer LNCaP cells, as well as their resistance to many apoptotic stimuli. The addition of bcl-2 antisense oligonucleotides, however, restored apoptosis. Our studies demonstrate gene silencing effects of the mRNA-cDNA transfection that is similar to those of PTGS/RNAi in this in vitro prostate cancer cell model. A potential RNA-directed RNA polymerase activity was also detected which is alpha-amanitin-sensitive. These findings indicate that a novel gene silencing system may exist in mammalian cells.

CONTROLLED TERM: Check Tags: Human
Base Sequence
Cell Line
DNA Primers
DNA, Complementary: GE, genetics
*DNA, Complementary: ME, metabolism

*Gene Silencing
*Genes, bcl-2
RNA, Messenger: GE, genetics
*RNA, Messenger: ME, metabolism
Transcription, Genetic
Tumor Cells, Cultured

CHEMICAL NAME: 0 (DNA Primers); 0 (DNA, Complementary); 0 (RNA, Messenger)

L3 ANSWER 4 OF 5 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 2001:234339 SCISEARCH
THE GENUINE ARTICLE: 409XJ
TITLE: A novel **mRNA-cDNA interference** phenomenon for silencing bcl-2 expression in human LNCaP cells
AUTHOR: Lin S L; Chuong C M (Reprint); Ying S Y
CORPORATE SOURCE: Univ So Calif, Keck Sch Med, Dept Pathol, HMR 209, 2011 Zonal Ave, Los Angeles, CA 90033 USA (Reprint); Univ So Calif, Keck Sch Med, Dept Pathol, Los Angeles, CA 90033 USA; Univ So Calif, Keck Sch Med, Dept Cell & Neurobiol, Los Angeles, CA 90033 USA; Epiclone Inc, Alhambra, CA 91801 USA
COUNTRY OF AUTHOR: USA
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (2 MAR 2001) Vol. 281, No. 3, pp. 639-644.
Publisher: ACADEMIC PRESS INC, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495 USA.
ISSN: 0006-291X.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 26
ABSTRACT:

The templates required for inducing posttranscriptional gene silencing (PTGS) effects have been investigated in human prostate cancer LNCaP cells. Transfection of a mRNA-cDNA hybrid construct was found to result in a relatively long-term interference of specific gene expression. Androgen-stimulated expression of bcl-2 has been reported to increase the tumorigenic and metastatic potentials of human prostate cancer LNCaP cells, as well as their resistance to many apoptotic stimuli. The addition of bcl-2 antisense oligonucleotides, however, restored apoptosis. Our studies demonstrate gene silencing effects of the mRNA-cDNA transfection that is similar to those of PTGS/RNAi in this in vitro prostate cancer cell model. A potential RNA-directed RNA polymerase activity was also detected which is alpha -amanitin-sensitive. These findings indicate that a novel gene silencing system may existinmammaliancells. (C) 2001 Academic Press.

CATEGORY: BIOCHEMISTRY & MOLECULAR BIOLOGY; BIOPHYSICS
SUPPLEMENTARY TERM: **mRNA-cDNA interference** phenomenon; (D-RNAi); posttranscriptional gene silencing (PTGS); RNA-directed RNA polymerase (RaRp); prostate cancer cells; bcl-2
SUPPL. TERM PLUS: DOUBLE-STRANDED-RNA; PROSTATE-CANCER CELLS; C-ELEGANS; IN-VIVO; MESSENGER-RNA; GENE-FUNCTION; APOPTOSIS; RESISTANCE; POLYMERASE; DROSOPHILA

REFERENCE(S):

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	ARN PG (RPG)	Referenced Work (RWK)
BAULCOMBE D C	2000	290	1108	SCIENCE
BERCHEM G J	1995	55	735	CANCER RES
BOSHER J M	2000	2	31	NAT CELL BIOL
COGONI C	1999	399	166	NATURE

COLOMBEL M	1993	143	390	AM J PATHOL
FILIPOVSKA J	2000	6	41	RNA
FIRE A	1998	391	806	NATURE
GRANT S R	1999	96	303	CELL
GRISHOK A	2000	287	2494	SCIENCE
HSIAO M	1997	233	359	BIOCHEM BIOPH RES CO
KETTING R F	1999	99	133	CELL
LIN S L	1999	27	4585	NUCLEIC ACIDS RES
LIN S L	1999	257	187	BIOCHEM BIOPH RES CO
MCCONKEY D J	1996	56	5594	CANCER RES
MISQUITTA L	1999	96	1451	P NATL ACAD SCI USA
MODAHL L E	2000	20	6030	MOL CELL BIOL
PALBHADRA M	1999	99	35	CELL
RAFFO A J	1995	55	4438	CANCER RES
REED J C	1990	50	6565	CANCER RES
SAMBROOK J	1989			MOL CLONING LAB MANU
SMARDON A	2000	10	169	CURR BIOL
TABARA H	1999	99	123	CELL
WARGELIUS A	1999	263	156	BIOCHEM BIOPH RES CO
WIANNY F	2000	2	70	NAT CELL BIOL
YANG D	2000	10	1191	CURR BIOL
ZAMORE P D	2000	101	25	CELL

L3 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:157362 CAPLUS
DOCUMENT NUMBER: 135:283853
ENTRY DATE: Entered STN: 06 Mar 2001

TITLE: A novel **mRNA-cDNA interference** phenomenon for silencing bcl-2 expression in human LNCaP cells

AUTHOR(S): Lin, Shi-Lúng; Chuong, Cheng-Ming; Ying, Shao-Yao
CORPORATE SOURCE: Department of Pathology, Keck School of Medicine, University of Southern California, Los Angeles, CA, 90033, USA

SOURCE: Biochemical and Biophysical Research Communications (2001), 281(3), 639-644
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
CLASSIFICATION: 3-4 (Biochemical Genetics)
Section cross-reference(s): 1

ABSTRACT:
The templates required for inducing posttranscriptional gene silencing (PTGS) effects have been investigated in human prostate cancer LNCaP cells. Transfection of a mRNA-cDNA hybrid construct was found to result in a relatively long-term interference of specific gene expression. Androgen-stimulated expression of bcl-2 has been reported to increase the tumorigenic and metastatic potentials of human prostate cancer LNCaP cells, as well as their resistance to many apoptotic stimuli. The addition of bcl-2 antisense oligonucleotides, however, restored apoptosis. Our studies demonstrate gene silencing effects of the mRNA-cDNA transfection that is similar to those of PTGS/RNAi in this in vitro prostate cancer cell model. A potential RNA-directed RNA polymerase activity was also detected which is α-amanitin-sensitive. These findings indicate that a novel gene silencing system may exist in mammalian cells. (c) 2001 Academic Press.

SUPPL. TERM: **mRNA cDNA interference**
silencing bcl2 human LNCaP; prostate cancer cell silencing bcl2 D RNAi
INDEX TERM: Animal cell line
(LNCaP, in vitro prostate cancer model; novel

mRNA-cDNA interference
(D-RNAi) phenomenon for silencing bcl-2 expression in human LNCaP cells)

INDEX TERM: Gene, animal
ROLE: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(bcl-2, silencing, by D-RNAi; novel **mRNA-cDNA interference** (D-RNAi) phenomenon for silencing bcl-2 expression in human LNCaP cells)

INDEX TERM: Prostate gland
(neoplasm, cells; novel **mRNA-cDNA interference** (D-RNAi) phenomenon for silencing bcl-2 expression in human LNCaP cells)

INDEX TERM: Antitumor agents
(potential; novel **mRNA-cDNA interference** (D-RNAi) phenomenon for silencing bcl-2 expression in human LNCaP cells)

INDEX TERM: Gene
(processes, similar to PTGS/RNAi, DNA-RNA interference, (D-RNAi), cDNA-mRNA hybrid; novel **mRNA-cDNA interference** (D-RNAi) phenomenon for silencing bcl-2 expression in human LNCaP cells)

INDEX TERM: 9026-28-2, RNA-directed RNA polymerase
ROLE: BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(RdRp-like enzyme for D-RNAi, α-amanitin-sensitive activity of; novel **mRNA-cDNA interference** (D-RNAi) phenomenon for silencing bcl-2 expression in human LNCaP cells)

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD.

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